To reviewer 1

Thank you for your useful comments. Based on your comments, we have revised the manuscript as follows:

1). address the issue of non-invasive testing such as ergonovine echocardiography etc. as an alternative or addition to invasive testing.

As you mentioned, several noninvasive assessments of coronary spasm have been reported to be useful, including ergonovine stress echocardiography; however, we have not been able to perform this at our institution. We have included this point in the "study limitation" section.

Fourth, noninvasive tests such as ergonovine stress echocardiography[30] and coronary computed tomography [31, 32] have been used to evaluate coronary spasm, and their usefulness has been reported; however, these tests are not routinely performed in our hospital and have not been evaluated.

2). report complication rates following coronary angiography, if there were any.

Based on your comments, we have added the following sentences in the "Methods" and "Results" sections.

In the "Methods"

Complications of CAG and SPT include common severe complications, such as myocardial infarction, cerebral infarction, vascular trauma requiring surgery

associated with CAG, induction of coronary spasm or coronary perforation associated with a pressure wire insertion, ventricular fibrillation (Vf), pulseless ventricular tachycardia (pVT) or hemodynamic compromise requiring catecholamine administration, and atrial fibrillation associated with SPT.

In the "Results"

No serious complications occurred with CAG, and no coronary spasm was induced with pressure wire insertion or coronary perforation was observed. In the SPT, no Vf or pVT occurred, but hemodynamic instability requiring catecholamine was observed in two patients (6%) in Group F and one patient (2%, p = 0.30%) in Group D. Atrial fibrillation during SPT was observed in four patients (12%) in group F and two patients (4%, p = 0.13) in Group D, but the difference was not statistically significant.

3). report whether any of the patients had myocardial bridging.

According to your suggestions, we have added the definition and frequency of myocardial bridging in the "Methods" and "Results".

In the "Methods" section

We also investigated the possibility of myocardial bridging (MB), which is the presence of a >20% systolic reduction in coronary artery diameter[23]

In the "Results" section

On CAG and SPT, the frequencies of coronary atherosclerosis (p = 0.03) and MB (p = 0.047, in table 3, p = 0.05) were significantly higher in Group F than in Group D, whereas the frequency of occurrence of multivessel spasm did not differ significantly between the two groups (Table 3).

To reviewer 2

Thank you for your useful comments. Based on your comments, we have revised the manuscript as follows:

This is an observational, retrospective study on using pressure wire during spasm provocation test in patients with VSA and exploring whether there is difference of intracoronary pressure among diffuse or focal spasm patients. Their results showed that a higher frequency of metabolic syndrome and coronary atherosclerosis in patients with VSA and focal spasm.
Major comments are below:

1. This clinical study is generally well done, the main concern is whether there is other non-invasive imaging strategy in evaluating the disease severity, for example CTA.

As you mentioned, several noninvasive assessments of coronary spasm have been reported to be useful, including coronary CT, but we have not been able to perform this at our institution, although a small number of patients with VSA with chest symptoms occurring on exertion underwent coronary CT. We have included this point in the "study limitation" section.

Fourth, noninvasive tests such as ergonovine stress echocardiography[30] and coronary computed tomography [31, 33] have been used to evaluate coronary

spasm, and their usefulness has been reported; however, these tests are not routinely performed in our hospital and have not been evaluated.

2. Authors discuss about the severity of myocardial ischemia caused by focal spasm stress, is there any direct clinical manifest data of myocardial ischemia, for example Troponin, CKMB level?

Thank you for your helpful comment. We measured troponin levels in patients with exacerbated chest symptoms or a long anginal attack duration, but the number of cases was too small for analysis. We have included this matter in the limitations of this study.

Fifth, we have experienced that troponin, a myocardial enzyme, is positive during severe myocardial ischemia in some VSA patients. We believe this is an important indicator for assessing the severity of myocardial ischemia, even in VSA patients. However, at our institution, it is not routinely measured in patients with worsening chest symptoms and a long duration of attacks.

3. Can authors discuss the fact that Calcium-channel blocker medication is significant in the focal spasm group?

Based on your comments, we have added the following sentences to the "Discussion" section:

In contrast, higher doses of ACh during spasm provocation appeared to be more frequently observed in focal spasms. In the present study, the frequency

of use of coronary vasodilators, such as CCB or long-acting nitrate, was high in the F group. Although we could not evaluate chest symptoms on admission in the present study, the high frequency of coronary vasodilator medications on admission may be due to the aim of improving chest symptoms due to the high degree of myocardial ischemia caused by focal spasm. At our institution, as per the guidelines [2], coronary vasodilators were discontinued 48 h prior to the SPT; however, the long-acting CCB may have remained effective, resulting in higher ACh doses in the SPT in the F group.

4. It appears that in the focal spasm group, there are more cases of high dose ACh inducing, does it cause certain bias in the p value?

Thank you for your valuable comments. The mechanism of different doses of ACh in the two lesions in the SPT is also discussed in Section 3. Although ACh dose was not a significant factor in the multivariate analysis, we included it in the study limitation because it may still have influenced the present results. Finally, the doses of ACh that induced the focal/diffuse spasms were different. Although this factor was not significant in the multivariate analysis, it may have influenced the reduction in intracoronary pressure during ACh provocation.

Minor comment: Table 1 Group S should be Group F

Thank you for your suggestion. We have changed “Group S” to “Group F”.